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# Consensus statement on crush injury and crush syndrome

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Crush syndrome remains rare in European practice. It is however common in areas of civil disorder and where the normal structures of society have given way to civil war or natural disaster. Western doctors are becoming increasingly involved in such situations and there is no reason to believe that instances due to more conventional causes, such as collapse in the elderly or road traffic accidents, will cease. For all these reasons it is important that clinicians who deal infrequently with crush syndrome have access to appropriate guidelines. This consensus report seeks to provide such advice. It reports the findings of a consensus meeting on crush injury and crush syndrome held in Birmingham on 31 May 2001, and coordinated by the Faculty of Pre-Hospital Care of the Royal College of Surgeons of Edinburgh.

**Key words:** crush syndrome; renal failure; natural disasters

## Introduction

Crush injuries and crush syndrome were first described in the English language literature by Bywaters and Beall (1941) after several patients who had been trapped under rubble of buildings bombed in the Blitz subsequently died of acute renal failure. It has been described in numerous settings since, most commonly after natural disasters such as earthquakes, in war, and after buildings have collapsed as a result of

explosion. Crush syndrome is also seen following industrial incidents such as mining and road traffic accidents. However, crush syndrome is not confined to traumatic aetiologies, and has also been described following periods of crush by patients' own body weight, after stroke or intoxication (Michaelson, 1992).

Most commonly in traumatic crush, the legs are affected, and less frequently the arms. Many authors believe that crush injury of the head and torso significant enough to cause the syndrome is incompatible with life due to the inherent internal organ damage, but there are a few reported cases of such instances (Hiraide *et al.*, 1997).

Crush syndrome bears many similarities to, but is distinct from, the syndrome caused by heat illness.

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Organisations represented: The Voluntary Aid Societies, The Ambulance Service Association, The British Association for Immediate Care, British Association for Emergency Medicine, Faculty of Accident and Emergency Medicine, The Royal College of Anaesthetists, The Royal College of Physicians, The Royal College of Surgeons of Edinburgh, The Intensive Care Society, The Royal College of Nursing, The Military, and The Faculty of Pre-hospital Care.

## Definition

Following a search of the literature, it was felt that a definition of crush injury and crush syndrome was required.

### Consensus view

'A crush injury is a direct injury resulting from crush. Crush syndrome is the systemic manifestation of muscle cell damage resulting from pressure or crushing'.

The severity of the condition is related to the magnitude and duration of the compressing force, and the bulk of muscle affected. The definition is not, however, dependent on the duration of the force applied.

Examples of this relationship include a patient whose legs are run over by the wheels of a truck. In this case the force is large, but the duration is very short. At the other extreme, there is the elderly patient who has suffered a stroke, falls, and lies in the same position for hours, sustaining a crush injury to the areas of the body on which they are lying. In this case, the force is relatively small, but crush syndrome may develop as a result of the prolonged period of pressure. Similar cases to this are described by Shaw *et al.* (1994) as a result of drug overdose.

### Pathogenesis and clinical features

The typical clinical features of crush syndrome are predominantly a result of traumatic rhabdomyolysis and subsequent release of muscle cell contents. The mechanism behind this in crush syndrome is the leakiness of the sarcolemmal membrane caused by pressure or stretching. As the sarcolemmal membrane is stretched, sodium, calcium and water leak into the sarcoplasm, trapping extracellular fluid inside the muscle cells. In addition to the influx of these elements into the cell, the cell releases potassium and other toxic substances such as myoglobin, phosphate and urate into the circulation (Better, 1999).

The end result of these events is shock (discussed below), hyperkalaemia (which may precipitate cardiac arrest), hypocalcaemia, metabolic acidosis, compartment syndrome (due to compartment swelling), and acute renal failure (ARF). The ARF is caused by a combination of hypovolaemia with subsequent renal vasoconstriction, metabolic acidosis and the insult of nephrotoxic substances such as myoglobin, urate and phosphate.

### Shock

Haemodynamic instability secondary to crush syndrome is multifactorial. First, many patients have

other injuries, such as fractures of the pelvis or lower limbs, sufficient in themselves to cause hypovolaemia. The sequestration of fluid into the affected muscle compartments has already been described, resulting in fluid shift from the intravascular to the intracellular compartments. This may cause hypovolaemia, as the intravascular volume is depleted. Electrolyte imbalances such as hyperkalaemia, hypocalcaemia and a metabolic acidosis will have a negatively inotropic effect, and there is also evidence that there is direct myocardial depression from other factors released when muscle cells are damaged (Rawlins *et al.*, 1999).

### Approach to treatment

Treatment of the crushed patient can be divided into two phases. The initial prehospital phase may, depending on the mechanism of injury, involve a prolonged extrication period. The second phase commences on reaching a definitive medical care facility.

In the case of prolonged on-scene time, or delay in transfer due to geographical reasons, some of the second phase guidelines may be employed in the pre-hospital environment.

### Consensus view

Safety is the first priority when approaching an accident scene, and this is particularly relevant to situations where patients may have suffered crush injuries, as there may be danger from falling debris or risk of further building collapse.

Once the scene has been declared safe, in cases of mass casualties a triage system, such as the triage sieve—Major Incident Medical Management & Support (MIMMS, 2002)—should be used to prioritize casualties and assess the need for further treatment. For each individual casualty, an assessment of airway, breathing and circulation is the next priority. Attention must be given in trauma to the possibility of spinal injury, and full spinal precautions should be maintained. Administration of high-flow oxygen by mask should be a priority in treatment, as should the arrest of any obvious external haemorrhage and the splinting of limb injuries. The patient should be exposed as necessary to assess and manage injuries. In a hostile environment or where there is a risk of hypothermia, exposure should be as limited as possible. Assessment of distal neurovascular status is essential if exposure is to be kept to a minimum.

## Fluid resuscitation

Once the initial primary survey has been performed, intravenous access should be obtained. If limb crush injury has occurred, and there is a likelihood of the patient developing crush syndrome, the following fluid guidelines should be followed. In the presence of life-threatening thoraco-abdominal injury, fluid resuscitation should be performed according to the Faculty's previously published guidelines (Greaves *et al.*, 2002).

### Consensus view

An initial fluid bolus of 2l of crystalloid should be given intravenously. This should be followed by 1–1.5 l per hour. The fluid of choice is normal saline, warmed if possible, as this is established as the fluid carried by the majority of prehospital vehicles in the UK. Hartmann's solution contains potassium and has a theoretical disadvantage of exacerbating hyperkalaemia. If possible, fluid should be started prior to extrication, however gaining intravenous access and the administration of fluid should not delay extrication and transport to a definitive care facility. Early catheterisation should be considered, especially if there is a prolonged extrication or evacuation phase.

Once the patient reaches hospital, 5% dextrose should be alternated with normal saline to reduce the potential sodium load.

## Analgesia

### Consensus view

The use of medical teams including paramedics, nurses and doctors should be considered at an early stage, and appropriate analgesia should be given. This may involve the use of Entonox<sup>®</sup> initially, but most patients will require intravenous analgesia such as an opiate, titrated against response. The use of ketamine, with or without the concomitant use of a benzodiazepine, is also an effective means of relieving pain, and may aid extrication.

## Triage

### Consensus view

Patients with crush injuries should be taken to a hospital with an intensive care facility and the equip-

ment and expertise necessary to provide renal support therapy such as haemofiltration or dialysis.

## Tourniquets

The use of tourniquets has a theoretical role in the management of these patients. If the release into the circulation of the contents of crushed muscle cells can be avoided, possibly with the use of a tourniquet, it may be of benefit. However, there is currently no available evidence to support this.

### Consensus view

The use of tourniquets should be reserved for otherwise uncontrollable life threatening haemorrhage. There is no evidence at the moment to support the use of tourniquets in the prevention of reperfusion injury following extrication, or in the prevention of washing of the products of rhabdomyolysis into the circulation.

## Amputation

Another theoretically advantageous measure is amputation of a crushed limb to prevent crush syndrome.

### Consensus view

There is no evidence to support the use of amputation as a prophylactic measure to prevent crush syndrome. Reports from the literature suggest that even severely crushed limbs can recover to full function. If the limb is literally hanging on by a thread, or if the patient's survival is in danger due to entrapment by a limb, amputation should be considered and appropriate expert advice sought.

## Immediate in-hospital care

### Consensus view

Patient should be assessed following normal Advanced Trauma Life Support (ATLS) 1997 guidelines. Baseline blood tests should be taken. These will include full blood count, urea and electrolytes, creatinine kinase, amylase, liver function tests, clotting screen and group and save (cross match if deemed appropriate). The patient should be catheterized and hourly urine measurements commenced. Central venous pressure and invasive arterial monitoring should be considered.

## The use of solute-alkaline diuresis

The development of acute renal failure in these patients significantly decreases the chances of survival (Ward, 1988). Every effort must be made, therefore, to prevent its occurrence. Alkalinisation of urine and the use of a solute-alkaline diuresis is accepted to be protective against the development of acute renal failure (Better, 1990, 1999).

### Consensus view

It is recommended that urine pH is measured, and kept above 6.5 by adding 50 mmol aliquots of bicarbonate (50 ml 8.4% sodium bicarbonate) to the intravenous fluid regime. Solute diuresis is affected by administering mannitol at a dose of 1–2 g/kg over the first four hours as a 20% solution, and further mannitol should be given to maintain a urine output of at least 8 l per day (300 ml/hour). Fluid requirements are high, usually of the order of 12 l per day, due to the sequestration of fluid in muscle tissue. Fluid should be given at approximately 500 ml/hour, but regular review of clinical parameters such as central venous pressure and urine output should dictate exact amounts of fluid given.

The maximum daily dose of mannitol is 200 g, and it should not be given to patients who are in established anuria.

## Children

### Consensus view

There is very little evidence in the literature to guide the treatment of children suffering from crush injuries. In young children the difference in body proportions, namely the reduced contribution to the total percentage made by the limbs, may influence the incidence of crush syndrome. The fluid resuscitation guidelines from Advanced Paediatric Life Support (APLS, 2001) of an initial bolus of 20 ml/kg should be followed in these patients.

## The elderly and patients with co-morbidity

### Consensus view

In the elderly, and those with pre-existing medical conditions such as cardiac failure, fluid replacement must be tailored to requirements and given with cau-

tion. Close monitoring of the clinical state of the patient, and regular review of fluid requirements is essential in these patients.

## Compartment syndrome

The development of compartment syndrome in crush injury is due to the uptake of fluid into damaged muscle tissue contained within the restricted compartment. Once compartment pressure exceeds capillary perfusion pressure at about 30–40 mmHg, the tissue inside the compartment becomes ischaemic, and compartment syndrome develops.

The traditional treatment of compartment syndrome is fasciotomy (Shaw *et al.*, 1994), but there is now evidence that initial treatment with mannitol may decompress compartment syndrome and avoid the need for surgery (Better *et al.*, 1991; Better, 1999).

### Consensus view

In patients with compartment syndrome due to crush injury, in the absence of neurovascular compromise, a trial of mannitol therapy should be instigated, but a specialist surgical opinion should be sought early.

## Hyperbaric oxygen therapy

There is theoretical and limited experimental evidence that hyperbaric oxygen therapy may improve wound healing and reduce the need for multiple surgical procedures in crush injury (Bouachour *et al.*, 1996).

High concentrations of oxygen cause systemic vasoconstriction but continue to deliver adequate oxygen delivery. In a similar fashion, nitric oxide synthase inhibitors may also have a role in preventing excessive vasodilatation in the crushed muscle and the consequent increase in third space fluid losses (Rubinstein *et al.*, 1998).

### Consensus view

Logistically hyperbaric oxygen treatment has limited application. Patients with no significant co-morbidity, and who can be managed in a hyperbaric chamber where the facility is available, may be treated with hyperbaric oxygen therapy. It is recommended that treatment options are discussed with the local hyperbaric unit. This is not recommended as first-line treatment. Patients should, however, receive high-flow oxygen, unless there is a specific contraindication.

## Further management

### Consensus view

In many cases, intensive care support will be required for the complications of crush syndrome. If the patient becomes oligo- or anuric, it is likely that they will require haemofiltration or dialysis.

## Multiple casualties

### Consensus view

In the civilian environment in the United Kingdom, there will be a huge strain on intensive care facilities if there are multiple crushed casualties. A policy should be drawn up to prepare for the dispersal of these casualties on both a national and international level should an incident occur. Further information is available in Better's review (1999).

## Areas identified for future research

### Use of tourniquets

Is there a role for the tourniquet post- or pre-extrication of the crush injury casualty? The use of an animal model of crush injury was suggested, to assess the suitability of tourniquet administration. Comparison of tourniquet placement versus no tourniquet in delayed intravenous fluid administration was suggested as a further research option. Are there any further deleterious effects due to the increased ischaemia times involved in application of a tourniquet?

Could cooling the limb be used in order to slow cellular respiration and consequently decrease oedema/compartment syndrome/improve limb viability?

Tourniquet effectiveness was highlighted as a potential problem with their use. There is a requirement to perform a literature search into tourniquet usage, in particular regarding their use in Biers blocks, in order to determine the effectiveness of certain types of tourniquet and the leakage rates of drugs, past the tourniquet. This may assist in establishing the likelihood of potassium leaking into the systemic circulation.

### Fluid administration

Types of fluid currently used for administration include: normal saline, Hartmann's, Dextran, or starches. What is the correct amount of fluid to be giving? Should we be looking at urine output, absolute

volume intake, or acidity of urine as a guide to fluid administration? Oedema occurring secondary to massive fluid administration may be detrimental. At what stage do we need to worry about this? What effect does this have on compartment syndrome?

### Prognostic indicators

Creatinine kinase, myoglobinaemia, and amylase have been suggested as prognostic indicators, although it is not clear that they can predict outcome at an early enough stage to allow effective intervention. The use of microalbuminuria as a prognostic indicator of crush syndrome was suggested.

### Hyperbaric oxygen therapy

Use of the Institute of Naval Medicine was suggested in order to evaluate the merits of this treatment modality. In view of the scarcity of this resource around the country it did not meet with a great deal of support.

### Bicarbonate administration

Early administration of bicarbonate intravenously is thought to decrease metabolic acidosis and promote alkalinisation of urine, which decreases the precipitation of myoglobin in the renal tubules. Administration of bicarbonate immediately post-extrication, in anticipated metabolic acidosis, was discussed. Has this been shown to be beneficial? Are there any detrimental features? What would be the appropriate and safe doses to use? Is there a role for the combined use of acetazolamide in order to prevent metabolic alkalosis following bicarbonate administration?

### Mannitol and compartment syndrome

There is anecdotal evidence in the literature that, owing to the high complication rate in performing fasciotomy for compartment syndrome in crushed patients, they are best managed with mannitol alone. It is suggested that there is a noticeable difference in diameter and symptoms of the lower leg within 40 minutes of administration of IV mannitol (Better, 1999). Fasciotomy should be reserved for refractory cases. The use of an animal model of a compartment syndrome is questioned due to the anatomical differences from humans. Many animals that are commonly used as models for humans, such as pigs, sheep and dogs do not have fascial compartments. Primates share similarities but, ethically, such experiments would be more difficult to justify. Further information on existing animal experimentation relating to compartment syndrome is required prior to planning any further projects.

## References

- Advanced Life Support Group.** 2001. *Advanced paediatric life support*, 2nd edn. London: BMJ Bookshops.
- American College of Surgeons Committee.** 1997. *Advanced Trauma Life Support for Doctors*. Chicago: American College of Surgeons.
- Better OS, Zinman C, Reis ND et al.** 1991. Hypertonic mannitol ameliorates intracompartmental tamponade in model compartment syndrome in the dog. *Nephron* **58**: 344–46.
- Better OS.** 1990. The crush syndrome revisited (1940–1990). *Nephron* **55**: 97–103.
- Better OS.** 1999. Rescue and salvage of casualties suffering from the crush syndrome after mass disasters. *Milit Med* **164**: 366–69.
- Bouachour G, Cronier P, Gouello JP et al.** 1996. Hyperbaric oxygen therapy in the management of crush injuries; a randomized double-blind placebo-controlled clinical trial. *J Trauma* **41**(2): 333–39.
- Bywaters EGL, Beall D.** 1941. Crush injuries with impairment of renal function. *Br Med J* **1**: 427.
- Greaves I, Porter KM, Revell MP.** 2002. Fluid resuscitation in pre-hospital trauma care: A consensus view. *J Roy Coll Surg Edinb* **47**(2): 451–57.
- Hiraide A, Ohnishi M, Tanaka H et al.** 1997. Abdominal and lower extremity crush syndrome. *Injury* **28**(9–10): 685–86.
- Major Incident Medical Management & Support (MIMMS).** 2002. London: BMJ Bookshops.
- Michaelson M.** 1992. Crush injury and crush syndrome. *World J Surg* **16**: 899–903.
- Rawlins M, Gullichsen E, Kuttala K et al.** 1999. Central hemodynamic changes in experimental muscle crush injury in pigs. *Eur Surg Res* **31**: 9–18.
- Rubinstein I, Abassi Z, Coleman R et al.** 1998. Involvement of nitric oxide system in experimental muscle crush injury. *J Clin Invest* **101**(6): 1325–33.
- Shaw AD, Sjolín SU, McQueen MM.** 1994. Crush syndrome following unconsciousness: need for urgent orthopaedic referral. *Br Med J* **309**: 857–59.
- Ward MM.** 1988. Factors predictive of acute renal failure in rhabdomyolysis. *Arch Intern Med* **148**: 1553–57.